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Bilateral Carotid Body Resection

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Public Health Service Assessment

Bilateral Carotid Body Resection as a Treatment for Chronic Intractable Bronchospastic Diseases

1985

INTRODUCTION

Bilateral carotid body resection (BCBR) is a procedure in which both carotid bodies are identified at surgery and excised for the purpose of relieving the sensation of shortness of breath. The procedure is performed on patients with asthma and chronic obstructive pulmonary disease (COPD), that is, chronic bronchitis and emphysema.

The carotid body is an oval, reddish-brown structure measuring 3 to 6 mm and located on each side of the neck on the posteromedial aspect of the common carotid artery at the superior portion of its bifurcation (1). Each carotid body is embedded within the carotid plexus and adventitia of the vessel wall. Microscopically, the parenchyma of the carotid body is formed by two types of cells: Type I cells are chemoreceptors ("glomus" or epithelial cells) that contain vesicles with an electron-dense core and have terminal nerves. Type II cells are supporting (sheath) cells that do not contain vesicles and are not innervated. The carotid sinus nerve contains afferent chemoreceptor and baroreceptor fibers and efferent sympathetic and inhibitory fibers (2). This nerve, a branch of the glossopharyngeal nerve, innervates the carotid body.

The carotid bodies play the major role in the initiation of the respiratory response to hypoxemia in man. Similar structures exist in the arch of the aorta, in the jugular vein, and in the pulmonary artery, but they do not significiantly affect the respiratory response. The carotid bodies function primarily as chemoreceptor reflex stations that

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discharge impulses to the reticular formation ("respiratory center") in the medulla oblongata (3). Chemoreceptors are sensitive to reduced arterial oxygen and to changes in carbon dioxide tension, temperature, and pH. They are able to respond to other forms of anoxia including that induced by drugs, such as nicotine and acetylcholine. Although respiration is ultimately controlled by the "respiratory center" in the brainstem, chemoreceptors primarily increase the rate, depth, and minute-volume of respiration in response to hypoxemia ("chemoreceptor respiratory drive") (4,5).

BACKGROUND

In 1961, Nakayama described a surgical treatment for the relief of asthma. He published a review of more than 3,900 cases which brought the procedure to the attention of the American medical community. Nakayama proposed that carotid body resection would "correct a reflex disturbance in the behavior of the bronchial system (6)." By 1963, Nakayama had reported on more than 4,000 patients who had been treated by unilateral carotid body resection (UCBR). In this series, 80 percent were reported "cured or improved" at 6 months after the surgery, and 58 percent maintained "good results" after 5 years (7). In 1962, Overholt, using a similar technique but including resection of carotid sinus nerves, reported on 157 patients with intractable asthma who had had a UCBR performed under local anesthesia (8). He hypothesized that removal of the carotid body would partially interrupt the pathway between the pulmonary plexus and the midbrain, thus dampening an overactive bronchoconstricting reflex. The results of this study were essentially the same as those reported by Nakayama: "Approximately four out of five intractable asthmatic patients were improved by carotid body removal." There was a gradual diminution of benefit to patients followed up to 30 months. The site of the resection, either right or left, was a matter of convenience. Neither of these large series reported histological verification of excised carotid body tissue. Early

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reports of successes following UCBR for patients with asthma or emphysema made this a popular technique for the treatment of severe obstructive airway disease. These enthusiastic reports, however, were later followed by studies showing less positive results.

In 1965, Segal and Dulfano reported on their study of 15 asthmatic patients who had had UCBR to relieve intractable bouts of coughing and wheezing (3). They found that the procedure afforded no significant clinical benefit, either immediate or delayed. All 15 patients returned to their chronically ill status, requiring intensive medication, within 1 month of surgery.

In 1968, a study from Finland reported on 29 patients ages 14 to 70 with intractable asthma or obstructive emphysema; 28 patients underwent UCBR, 1 underwent BCBR. Histology was positive for carotid body tissue in 25 of the 29 cases (9). Followup in 27 cases was from 6 to 41 months. The results indicated that 9 patients were free of asthma attacks, 1 patient was slightly improved, 6 were unchanged, and 11 were worse. Pulmonary function tests and acid-base balance were not found to correlate in any way with subjective symptoms following surgery. The authors concluded that benefit to patients was entirely subjective and that baseline symptoms returned to most patients in the course of time.

In 1965, Sedwitz et al., at the Mayo Clinic, reported on UCBR performed on 12 patients with poorly controlled asthma (10). Pulmonary function studies were performed before and after surgery. After a followup of at least 1 year, the clinical course and pulmonary function tests were unchanged in all patients. No patient was able to discontinue the use of steroids. Histology was positive for carotid body tissue in 9 of the 12 cases.

In a randomized study of UCBR reported in 1966, Curran et al. evaluated 23 patients with severe asthma, all of whom had had a minimum of 6 months of followup (11). A striking degree of subjective improvement in 13 patients who had sham

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operations led to the conclusion that the major effect of UCBR was probably psychological. The other 10 operated cases had histologic verification of carotid body tissue.

A randomized controlled trial of UCBR involving eight patients in the mid-1960's was performed at UCLA by Marschke et al. (12). After 6 to 12 months, virtually all patients in both the treated and control groups felt that they had benefited from the operation. Their physicians concurred with this opinion, and pulmonary function tests confirmed these subjective judgments. The authors suggested that some of the improvement may have been due to the placebo effect of a new, well-publicized, and allegedly successful procedure. There was no evidence to suggest that an abnormal carotid body reflex had been abolished.

In 1964, O'Rourke and O'Rourke reported on their evaluation of 72 asthmatics who underwent UCBR (verified histologically in 70 of 72 cases) and 17 asthmatic patients who had sham operations (13). They found that 42 of the 72 "operated" patients and 11 of the 17 "sham operated" patients derived benefit from the procedures. They also concluded that the favorable response to the surgery was probably psychological.

Interest in carotid body resection revived in the 1970's as a result of Winter's development of a surgical technique that involved bilateral resection of the carotid bodies without denervation of the carotid sinuses (14). This procedure, which avoids producing baroreceptor dysfunction, was said to produce symptomatic improvement in patients with asthma and emphysema despite a blunted chemoreceptor response to hypoxemia (15).

RATIONALE

The precise way BCBR relieves pulmonary symptoms in patients with asthma and other obstructive airway diseases is not clear. Some suggest that it appears to involve

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interruption of the sympathetic and parasympathetic input, which dampens the hyperactivity of the chemoreceptor bronchoconstrictor reflex system. This physiological alteration is thought to be a raising of the threshold for activating the reflex system by preventing the triggering of the respiratory reflex, thereby averting bronchoconstriction. Proponents argue that the reduction in airway resistance that follows BCBR permits clearing of bronchial secretions and is associated with clinical improvement (16,17).

Hypoxemia has been shown to result in increased airway resistance (bronchoconstriction) in animals, by Nadel and Widdicombe, and in man, by Astin and Penman (18,19). The results of various studies with regard to increased airway resistance in humans are conflicting. Some investigators have found decreased airway resistance or no change (20). Advocates of BCBR, however, have proposed the ability of the procedure to dampen chemoreceptor reflex responses as the rationale for the use of this procedure in patients with COPD. The expected benefits of BCBR include the loss of the sensation of breathlessness, diminished spontaneous cough, loss of wheezing, loss of exercise-induced bronchospasm, diminished air-trapping, and increased exercise tolerance. (15).

REVIEW OF THE LITERATURE

In 1972, Winter reported on 18 patients who had not benefited from a UCBR despite histological verification of carotid body tissue (14). The group included patients under age 40 who were "probably asthmatics" and patients over age 40 who were thought "likely" to have COPD with a major asthmatic component. BCBR performed in this group of patients, when reevaluated 6 months postoperatively, yielded success (marked or significant improvement) in 78 percent and equivocal results in 22 percent. Morbidity included transient postural hypotension in 3 older patients and nonspecific transient headaches in most patients.

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In a study of 14 cases of intractable asthma in children (under age 16) who had had BCBR, Winter reported, in 1981, that all patients became asymptomatic in the immediate postoperative period, and 6 patients remained asymptomatic after long-term followup (15). Of these, 1 remained asymptomatic during 10 years of followup; 5 others who were asymptomatic for 3 to 7 years were lost to further followup. Eight patients developed recurrent asthma within 30 days of surgery, but their symptoms were more readily controlled than prior to BCBR and gradually disappeared within 6 months to 5 years postoperatively.

Hyperpnea normally occurs as a response to hypoxemia; this ventilatory response is eliminated in patients after BCBR. In 1978, Chang et al. reported on one asthmatic patient who had undergone BCBR and had experienced no subjective discomfort during asthmatic attacks even when these episodes resulted in cyanosis and disorientation requiring emergency hospitalization (21). The authors compared this patient with six other asthmatic controls whose carotid bodies were intact. In breath-holding tests, the patient's breath-holding time was approximately twice as long as the mean time of the controls. The authors concluded that BCBR in asthmatic patients is hazardous, because of the risk of repeated episodes of hypoxemia.

In a review of carotid body resection in 1973, Winter reported the results of BCBR performed in 143 patients, as follows (22):

Number of patients who underwent BCBR	Results			Reference
	Improved	Unimproved	Worse	
4	2	2	-	Keim (23)
16	4	12	-	Sedwitz et al (10)
3	1	-	2	Wood et al (24)
87	52	29	6	Takino and Takino (25)
15	10	5	-	Phillips (26)
18	16	2	-	Winter (14)
Total 143	85 (60%)	50 (34%)	8 (6%)	

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In a group of 135 patients, of whom 124 were diagnosed as having COPD and 11 as having asthma, evaluations were performed pre and post BCBR. Winter performed perfusion scans of the lungs (27). He demonstrated that by 1 year postoperatively, 48 percent exhibited signs of improvement, 45 percent showed no change, and 7 percent were worse.

Of 87 patients who underwent BCBR and were followed by Takino and Takino, 52 were completely relieved of symptoms at first. However, the asthmatic attacks recurred within 18 months, except for 1 case, in which the attacks recurred after five years. These authors noted that although the temporary effect of BCBR may have appeared dramatic, all asthmatics usually relapsed (most within 2 years and the longest at 6 years.)

Seven patients, ages 16 to 48, who had a history of asthma but who had become asymptomatic following BCBR, were studied by Lugliani et al (4) at UCLA. They demonstrated that the ventilatory response to hypoxemia was abolished by the procedure. Furthermore, the response to increased arterial carbon dioxide was reduced 30 percent, compared with asymptomatic asthmatic controls. In these subjects, metabolic acidosis induced by exercise was not corrected by the expected compensatory mechanisms. Measurements of ventilation while breathing air at rest or during steady-state exercise showed no differences between subjects and controls.

Davidson et al. studied five patients who had BCBR (28). Breath-holding time averaged twice that of normal subjects and asthmatic "controls." These findings suggest that the carotid bodies contribute to the sensation of breathlessness and that reduced breathlessness after BCBR might be due to the removal of stimuli originating from these chemoreceptors.

Exercise tolerance studies in patients with chronic airway obstruction suggest that BCBR removes a component of the drive to increase ventilation during exercise (29). A loss of the ventilatory response during exericse following BCBR permits greater work tolerance and exercise for a short duration, but at the cost of increasing hypoxemia and acidosis.

Honda et al. compared exercise hyperpnea in five asthmatics 25 years after BCBR

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with 12 matched controls (30). They found that ventilation rose less and pCO_2 was higher in the post-BCBR group than in the controls. Because of less tachypnea, the operated patients had increased exercise tolerance.

A recent unpublished Belgian report on 26 patients following BCBR demonstrated that the mean preoperative pCO_2 increased from 44.3 to 50.5 mm Hg following surgery (31). The mean pO_2 fell from 67.9 to 59.6 mm Hg. Data on long-term followup are not available, but approximately three-quarters of the patients appeared to get relief from dyspnea. These findings suggest that BCBR may be associated with worsening of preexisting hypoxemia and primary respiratory acidosis.

DISCUSSION

The evaluation of BCBR in diseases such as asthma, which are subject to spontaneous exacerbations and remissions, is difficult. This difficulty is compounded by uncertainty about the comparability of the groups studied, differences in operative technique, failure to obtain histological confirmation of excised carotid body tissue, and lack of uniform measures of improvement.

Case reports indicate that the ventilatory drive in response to hypoxemia is lost or reduced in patients following BCBR. These patients can hold their breath significantly longer than controls with intact carotid bodies. Evidence is lacking that their underlying disease is improved or that there is reversal of respiratory obstruction. On the contrary, in patients with COPD, the reduction in ventilatory drive by removal of the carotid bodies results in an elevation of pCO_2 and depression of pO_2 (30). In addition, BCBR, by decreasing ventilatory drive, may be associated with irreversible effects as a result of increased pulmonary insufficiency (32).

The benefits of a reduced sensation of dyspnea and improved exercise tolerance following BCBR are offset by the inability to respond to hypoxemia. Tai and Read have shown that dangerous levels of hypoxemia may develop rapidly when asthma is exacerbated (33). In the absence of carotid bodies, reduced oxygenation may occur

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without warning symptoms. The diminished sensitivity to hypoxemia increases the risks for respiratory depression and death, which have been widely underestimated in association with chronic lung disease (34).

Sullivan investigated the ventilatory and waking responses to hypoxemia during sleep in dogs (35). He found that hypoxemia is a powerful arousal-promoting stimulus which, like the ventilatory response, appears to be mediated by the carotid bodies. Other investigators have studied cats but have not been able to demonstrate that a hypoxic arousal response was mediated by the carotid bodies (36). The loss of this response may be another potential hazard of BCBR, because it increases the vulnerability of patients with asthma or COPD to hypoxic death during sleep.

Carotid body resections do not have widespread clinical acceptability and have not been critically assessed in a controlled clinical trial. In 1968, the American Thoracic Society (in a publication titled "Current Status of the Surgical Treatment of Pulmonary Emphysema and Asthma") stated that "until better evidence on improvement is presented, this procedure (unilateral carotid body resection) has no place in the treatment of patients with asthma or emphysema."

In 1977, the National Institutes of Health (NIH) established a task force to assess the clinical effectiveness of BCBR. This task force carried out an extensive review of the published literature. It also obtained information from physicians performing the procedure and from those responsible for the postoperative care of the patients. The task force concluded that the clinical effectiveness of BCBR has not been demonstrated and that, even in the hands of experienced surgeons, the operation carried an in-hospital mortality of approximately 6 percent (26 out of 430) which is unacceptable for an elective surgical procedure (20).

A Federal Register notice of this assessment was published in October, 1984 (37).

In February, 1985 the NIH has informed the Office of Health Technology Assessment that neither the effectiveness of BCBR nor the risks incurred by the absence

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of both carotid bodies have been critically assessed in controlled clinical studies. Controlled physiological studies performed on patients following BCBR indicate elevated pCO_2 and decreased pO_2 levels, compared with controls, and weak or absent ventilatory responses to hypoxia. This situation could be life-threatening in patients with COPD, who frequently have hypoxemic episodes during sleep, exercise, or exacerbations of their disease. NIH concluded that the risks of the surgery itself and the additional risks of hypoventilation and the inability to respond appropriately to hypoxia appear to be unacceptable.

SUMMARY

Bilateral carotid body resection (BCBR) is a procedure in which both carotid bodies are surgically excised for the purpose of relieving the symptoms of COPD. A surgical treatment for the relief of asthma employing a unilateral carotid body resection was first performed in Japan in 1941, and thousands of the procedures were performed worldwide over the next two decades. In the mid-1960's, several controlled studies indicated that the procedure was ineffective in relieving the symptoms of these patients. It was suggested that the major effect of the surgery was probably "psychological." Interest in carotid body surgery was revived as a result of the work of Winter, who developed a technique of bilateral carotid body resection without denervation of the carotid sinuses. This procedure appeared to produce symptomatic improvement in patients with COPD despite a blunted chemoreceptor ventilatory response to hypoxemia. However, the clinical efficacy of the procedure in treating COPD has not been documented in any controlled trial. The risks of the surgery itself, and the potential for life-threatening episodes due to a loss of the ventilatory response to hypoxemia, suggest that this procedure may be hazardous and should not be regarded as safe and effective until data from properly controlled studies indicate otherwise.

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16. Abstract (Limit: 200 words)

Bilateral carotid body resection (BCBR) is a procedure in which the carotid bodies are excised for the purpose of relieving dyspnea. The procedure is performed on patients with asthma or chronic obstructive pulmonary disease (COPD). The carotid bodies function as chemoreceptors which reflexly increase respiration in response to hypoxemia. The clinical efficacy of BCBR in the treatment of COPD has not been documented in controlled trials. In a number of uncontrolled trials approximately 60 percent of patients obtained "improvement" following BCBR, while 34 percent were "unimproved" and 6 percent were "worse". Although case reports indicate that the ventilatory drive in response to hypoxemia is lost or reduced in patients following BCBR, evidence is lacking that their underlying disease is improved or that there is reversal of respiratory obstruction. In addition, the diminished sensitivity to hypoxemia increases the risks for respiratory depression and death. Carotid body resection carried an in-hospital mortality of approximately 6 percent. The procedure has not achieved widespread clinical acceptability. The surgical risks and the additional risks of hypoventilation and the inability to respond appropriately to hypoxemia following BCBR suggest that the procedure should not be regarded as safe and effective.

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